therapeutic choice because the mortality is lower than that of surgical treatment and its application is easy and rapid.[1,2]

We do not know with certainty how long it takes thrombolytic therapy to deocclude a thrombosed prosthesis, although it probably takes less time than surgery because of all the equipment needed to implement aggressive treatment.

The great risk of a redo valve replacement in these generally critically ill patients is also widely appreciated. The main risks of thrombolytic treatment are the thromboembolic complications, which appear in from 4% to 13% of the patients, and bleeding, which occurs in from 1.4% to 5%.[3]

We have read with interest the excellent report written by Nguyen and collegues.[4] They have added an important case to the medical literature for the successful application of the thrombolytic protocol with recombinant tissue-type plasminogen activator (rt-PA), which has not been used previously in the management of PVT. It consisted in a continuous intravenous infusion of rt-PA at a rate of 1 mg/h together with the administration of heparin in a continuous intravenous infusion of $3\ U \cdot kg^{-1} \cdot h^{-1}$. The duration of treatment was 80 hours. At the end of the fibrinolytic infusion, the transprosthetic gradients had decreased from a peak and mean of 158 and 86 mm Hg to 48 and 25 mm Hg, respectively. Fluoroscopy confirmed normal motion of the prosthetic valve. The patient's symptoms resolved.

We would like to make some comments related to this therapeutic regimen. Treatment with rt-PA in PVT has not been widely used. It has been blamed for a major risk of embolism other than thrombolysis for its potential and velocity of the infusion. Shapira and collegues[5] proved the efficacy and safety of rt-PA, with the additional advantage that if the thrombolytic treatment fails, surgery can be used with less risk for its less lytic systemic effect.

The regimen of administration is not well defined. This protocol probably needs a longer course and lower dose to provide better thrombolytic efficacy with less risk of complications in hemodynamically stable patients, because they do not need a prompt thrombolytic effect. An accelerated protocol with rt-PA should be reserved for critically ill patients.

Until now, streptokinase is the most effective thrombolytic agent used, alone or as a part of a sequential fibrinolytic treatment in the PVT.

Despite the favorable evidence of thrombolytic therapy in the treatment of the PVT, more data should be gathered to obtain a general consensus of the ideal management of this complication.

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Optimizing selective cerebral perfusion in adult aortic arch repair: Clinical relevance of the laboratory model

To the Editor:

I read with great interest the excellent article by Halstead and colleagues[1] detailing in their porcine model of deep hypothermic circulatory arrest (DHCA) the neuroprotective effects of selective cerebral perfusion (SCP) via both carotid arteries at a mean of 50 mm Hg for a period of 90 minutes. In this laboratory model, the authors have clearly demonstrated the adverse cerebral effects associated with SCP at higher pressures and flow rates. The clinical relevance of this observation is illustrated in the study by Khaladji and colleagues,[2] in which they analyze outcomes after hypothermic circulatory arrest (71.1% hemiarch; 10.4% total arch) and bilateral cold selective SCP at a perfusion pressure of 40 to 60 mm Hg with flow rates of 400 to 650 mL/min.

However, Halstead and colleagues chose a long SCP time of 90 minutes, which is the time required for a total arch repair. In an earlier clinical study, these investigators[3] demonstrated their technique with a trifurcated graft with mean DHCA/SCP times of 31.1 $\pm$ 6.6 minutes and 65.3 $\pm$ 20.9 minutes, respectively, with SCP perfusion pressures of 50 to 70 mm Hg with flow rates of 800 to 1200 mL/min. Hence, this latest laboratory study is part of their ongoing quest to optimize their technique of total arch replacement with SCP, and it suggests a new range for bilateral SCP perfusion pressures and flow rate.

However, although this model is clinically relevant for hemiarch repairs,[2] how might it apply in the case of aortic arch repair with unilateral SCP?[4] Would lower SCP perfusion pressures be clinically superior, assuming a clinically competent circle of Willis? Or would the contralateral brain be at significant risk of ischemia, given the relevant incidence of clinical inadequacy in the circle of Willis for cerebral perfusion in DHCA with unilateral SCP?[5] Do the authors plan to evaluate unilateral SCP in their porcine DHCA model?

I congratulate the authors again on their important contribution. I look forward to their comments about these aspects of selective cerebral perfusion during adult aortic arch repair.

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